International Journal of Medicine and Pharmaceutical Sciences (IJMPS)
ISSN(P): 2250-0049; ISSN(E): 2321-0095
Vol. 3, Issue 5, Dec 2013, 49-56

SEROPREVALENCE AND RISK FACTORS FOR HELOCOBACTER PYLORI INFECTION IN EAST MALAYSIAN COMMUNITY

MUSTAFA MURTAZA[1] & MENON JAYARAM[2]

[1]School of Medicine, University Malaysia Sabah, KotaKinabalu, Sabah, Malaysia

[2]Department of Gastroenterology, Hospital Queen Elizabeth, KotaKinabalu, Sabah, Malaysia

ABSTRACT

Background

Recent studies have shown that *Helicobacter pylori* infection proved to be of great public health importance in the developing countries and developed countries, especially in low socioeconomic groups. Poor hygiene suboptimal sanitation and crowded conditions; have been reported as risk factors.

The Aim of the Study was

To investigate the sero-epidemiology of *H. pylori* infection in the three districts of Sabah e.g. Kota Kinabalu, Kota Marudu and Kudat.

Methods

The cross sectional study describes the prevalence of infection with *H. pylori* as determined by a serodiagnostic assay.

Results

A prevalence of (32.26 %) in 1156 Subjects, in age groups 12 to 80 years from three districts, using a common protocol for blood collection and serological testing. In all populations combined, the prevalence of infection was higher in the ethnic Kadazan community 76.1%(P<0.05) compared with Malay, Bajau (14.2%) community. Infection was higher (59.1%) in the women than in men (41 %).Lower income groups had significantly higher prevalence 76.2% (P<0.01). Older age groups had higher infection (42.8%) than the younger age groups. Subjects with higher education had lower levels of infection ((27.9 %) compared with subjects with education up to primary education 36.6%(P<0.01),groups with secondary education, (17.3%),subjects with no education(18.4%).Increasing prevalence of infection in subjects with increasing body mass indexed (44.3%), compared with normal weight (35. 8%). There was considerable effect of smoking and alcohol consumption on the prevalence of infection. Former smokers had infection 79.8 %(P<0.05) compared to current smokers (5.5%). Former alcohol consumers had infection (79.8 %) compared to no-alcohol consumer (14.7%).

In Conclusion

There was considerably variation in the prevalence of infection within populations ethnicity, socioeconomics, and lower education levels was consistently associated with prevalence of infection.

KEYWORDS: *Helicobacter pylori,* Sero-Epidemiology, EthnicKadazan, East Malaysian Community

INTRODUCTION

Helicobacter pylori discovered in 1983, (1,2) a bacterium that infects one half or more of world population [3] proved to have profound public –health implications(4,5) *H.pylori* causes chronic gastritis, peptic ulcer and gastric cancer

(6,7).Chronic gastritis and peptic ulcer disease are common among the elderly and low-income groups (8).. There is clear link between *H pylori* infection and peptic ulcer (6).It has been estimated that 10 -20 % of *H .pylori* infected persons will develop a peptic ulcer in their life time (9). The relationship between *H. Pylori* and gastric cancer was discovered. Studies in Europe, Latin America, China, and Japan aim to determine whether the. *H. pylori* elimination prevents carcinoma or progression of precancerous lesions (10).

The high frequency of gastric cancer in Asia led to their widespread screening program. Most studies examining the association between *H.pylori* gastric cancers have focused on older age patients when *H.pylori* prevalence is more common. Haruma *et al* examined the frequency of *H.pylori* in patients with gastric cancer who were younger than 30 years of age (11). Helicobacter pylori is detectablele in nearly 95-100% of adult patients with duodenal ulcer and about 80% of patients with gastric cancer (12).The suspected association between gastric ulcer and gastric cancer is now being confirmed (13). In developing countries where most children become infected by the age of 10, gastric cancer rates are very high (14).

Lower socioeconomics, poor hygiene standards, crowded households and deficient sanitation are important to both acquisition of infection in childhood and spreading of the disease within the household (15, 16). Malaysia a multiracial country with three main ethnic races e.g. Malay, Indian and Chinese in west Malaysia and in East Malaysia, Sarawak and Sabah there are various indigenous ethnic groups. The highest prevalence rate was recorded among indigenous races in Kota Kinabalu, Sabah (17). Epidemiological data from Malaysia have repeatedly confirmed a low prevalence of *H.pylori* infection in the Malays compared to Indian and Chinese (18).Sabah has approximately 40 ethnic races, Kadazan, Dusun, Bajau and Brunei Malays are predominant groups. Sabah in terms of population is most populous state in Malaysia. The present study was carried out to investigate the prevalence of *H.pylori* infection and risk factors, according to ethnicity and gender among the three districts of Sabah.

METHODS

Study Subjects and Sample Collection

Three districts of in Sabah, Kudat, Kota Marudu and Kota Kinabalu with combined population of 650000 was selected. The study was conducted in outpatient clinics at Kudat District hospital, Kota Marudu District hospital and Luyang Clinic, Kota Kinabalu. Subjects of ages between 12 years to 80 years were included in the study. Subjects suffering from *H.pylori* infection, on active treatment for *H.pylori* and past treatment for *H.pylori* infection were excluded The subjects were required to answer the previously validated Leeds Dyspepsia Questionnaire (LDQ) Informed consent was obtained from the subjects. Demographic details of all subjects were recorded in the study protocol. Ethic clearance for the conduct of study was obtained from the Ministry of Health Malaysia.

Methods

A 5ml blood samples were processed and analyzed on *H.pylori*IgG immunoassay (ELISA).Test measured immunoglobulin IgG. Locally validated *H.pylori*IgG EIA kit 96 T(General Biological Corporation, Taiwan) was used throughout the project. *H.pylori* EIA index less than 0.90 was seronegative to IgG antibody *to H.pylori* and *H.pylori*IgG index of I.00 or greater was considered positive. Data was analyzed using Statistical Package for Social Sciences (SPSS-I6).The differences between values were considered significant when $P<0.05$ was obtained.

RESULTS

In total, number of 1200 subjects was recruited in the study and 1156 samples were assayed for the presence of Anti-*H.pylori*IgG antibodies. Forty four subjects were excluded for incomplete information Seropositivity was foundin 41

% male (176/429) and 59%(430/727) in the female subjects (Table..1) .Age had profound effect on beingseropositive. age group 50-59 years and >60 years had most seropositivity but lower seropositive was detected in the subjects in age <20 years (Table1). The prevalence of *H.pylori* was significantly higher 76.1 %(p<0.05) in the ethnic Kadazan/ Dusun community than the ethnic Malay, Bajau, ,community 14.2 % and Chinese 10.0 % (Table1) .Overweight, and normal weight subjects had prevalence 44.3% and 35.8%,and the obese subjects,15.5% seropositive respectively. Former smokers had high prevalence of 79.8 %,(p<0.05) compared to subjects with current smoking habit, 5.5% and never smoking 14.2%.Former alcohol consumers' subjects had higher prevalence of 79.8% compared to non-alcohol consumer (14.2%), and subjects' current alcohol consumer (5.5%). Lower income group had significantly higher prevalence (76.2%) compared to high income group (23.6 %)(Table. 1).Education level also had marked difference in prevalence, group with primary education 36.6 %,(p<0.01) group with secondary education, (17.3%),group with no education level, (18.4%),and with higher education (27.9 %), respectively (Table.1).

Tablele 1: Prevalence of *H.pylori*seropositivity by Categories of Independent Variables. (n=1156)

	IgG Category			
Age Category	**Negative**	**Equivocal**	**Positive**	**Total**
<20	200 (70.0%)	20 (7.0%)	66 (23.0%)	286
30-39	154 (62.0%)	18 (7.0%)	78 (31.0%)	250
40-49	146 (55.0%)	26 (10.0%)	92 (35.0%)	264
50-59	118 (54.0%)	21(9.5%)	80 (36.5%)	219
>60	67(48.5)	12 (8.7%)	58 (42.8%)	137
Gender				
Male	215 (50.1%)	38 (8.9%)	176 (41.0%)	429
Female	252 (34.7%)	45 (6.2%)	430 (59.1%)	727
Ethnic Category				
Malay, Bajau, Bugis, Brunei, Kadayan, Suluk, Bisaya, Jawa, Banjar,	186 (80.1%)	13 (5.6%)	33 (14.2%)	232
Chinese	156 (82.5%)	14 (7.5%)	19 (10.0%)	189
Kadazan, Dusun, Sino, Murut, Sungai, Rungus	156 (21.3%)	19 (2.6%)	560 (76.1%)	735
BMI Category				
Underweight	52 (72.2%)	17 (23.6%)	3 (4.2%)	72
Normal	238 (59.5%)	19 (4.7%)	143 (35.8%)	400
Overweight	243 (58.6%)	58 (10.8%)	240 (44.3%)	541
Obesity	95 (66.4%)	26 (18.1%)	22 (15.5%)	143
Smoking				
No	94(60.2%)	40 (25.6%)	22 (14.2%)	156
Ex-Smoker	130 (13.7%)	61 (6.5%)	755 (79.8%)	946
Smoking	33 (61.1%)	18 (33.4%)	3 (5.5%)	54
Alcohol Consumer				
No	109 (70.0%)	24 (15.3%)	23 (14.7%)	156
Ex-Consumer	130 (13.8%)	61 (6.4%)	755 (79.8%)	946
Yes	33 (61.1%)	18(33.3%)	3 (5.5%)	54
Income Group				
> 1000	282 (71.4%)	20 (5.0%)	93 (23.6%)	395
< 1000	142 (19.0%)	38 (4.8%)	580 (76.2%)	761
Education Level				
Nil	101 (60.2%)	36 (21.2%)	31 (18.4%)	168
Primary School	289 (57.8%)	28 (5.6%)	183 (36.6%)	500
Secondary School	194 (75.0%)	20 (7.7%)	45 (17.3%)	259
Higher Education	120 (52.4%)	45 (19.6%)	64 (27.9%)	229

DISCUSSIONS

Helicobacter pylori a microorganism infecting one half of the world population .*H .pylori* infection is common in adults, infants and children. The etiological role of this infection is accepted in peptic ulcer disease and gastric malignancy. The humans are the principal reservoir. The transmission occurs via person –to- person passage, and unclean water implicated in infection transmission it is well demonstrated that the prevalence of *H.pylori* infection varies between developing countries and developed countries, where prevalence rate among adults is high. Moreover, modes and risk factors of transmission, as well as reinfection rates are likely to vary between developing countries and developed countries (19, 20). The socioeconomic factors are important in *H.pylori* epidemiology. Socioeconomic factors such as low income, high household density of children, lack of clean water and poor sanitation were found to be important factors. Low socioeconomic factors likewise constitute a main risk factor in many developing countries (21)Many reports suggest declining frequency of the infection, particularly in those regions with higher or rapidly improving socioeconomic conditions. Rosendaal*et al*. studied the seroprevalence of *H. pylori* in the Netherlands in children (6-8 years of age n=154) and young adolescent (12-15 years of age n=160) in 1993 and compared them to samples collected in these same age groups in 1978. *H.pylori* prevalence declined from 19 % to 9.0 % in the 6- 8 year age group and from 23.0 % to 11.0 % in adolescent group (22). The decreasing frequency of *H.pylori* infection has been observed in many countries, but it is not universal. The infection remains common in almost all developing countries and in subpopulations. Overall *H.pylori* prevalence in the USA is estimated at 30 – 40 %, but it remains much higher in ethnic groups such as African-Americans and Hispanics (23). As observed in our study 76.4 % prevalence in lower income group, which is consistent withother studies (19,20). The prevalence of *H.pylori* infection is chiefly related to age and geographic location. In developing countries, by age 10 more than 70 % carry *H.pylori* and by age 20, carriage is nearly universal (24,25).In the United States, among non-Hispanic whites, little colonization occurs during childhood and rates gradually increase during adult hood and reach prevalence of 50 % among person older than 60 years (26).Among African –American and Hispanics, a higher prevalence is seen at all ages (27).The annual incidence of acquisition has ranged from 0.5 % among epidemiologist in the United States to 7.4 % among persons at an institution for the mentally retarded in Australia. (28).The older subjects were born at a time when the risk of infection in childhood was higher than those born later and therefore the high prevalence in elderly people should reflect their greater exposure to infection in their early years (29,30). Our results showed a high prevalence of *H. pylori* infection, and strong relation with age. A prevalence rates of 36.5% among 50 -59-age group and 42.8% among over 60 yearsage group.

In a study on Alaska Native residents, over 2000 serum samples were collected in 13 regions were assayed for *H.pylori*IgG antibodies. Seventy eight per cent of the residents were tested with an overall positive prevalence of 75.0 %; 40 % of children were seropositive by 4 years of age and 70 % by 10 years of age (31).

.Another study on ethnic African-Americans and Hispanics shows higher prevalence compared to White American (26).In our study *H.pylori*infection prevalence is higher 76.1 % in the ethnic Kadazan /Dusun community as compared to ethnic Malay, Bajau, 14.2 %,and 10% in Chinese, consistent with other studies. Other researchers have shown that*H. pylori* infection prevalence in males and females have equal rates of colonization (slight male predominance), (24). In another study found no difference in prevalence of infection between men and women (32). In this study a female predominance 59.1% compared to 41.0% male prevalence, contrast to other studies. Only explanation could be in our study more females were recruited.

Eurogast study group has shown an association between *H.pylori* infection and body mass index (BMI). In contrast with the effect of education standard, the effect of BMI was most pronounce in the younger age group.

Possibly BMI at a younger age reflects more accurately the relevant social class related aspects of current living conditions than does BMI at older age. Alternatively, BMI at older age may be more dominated by random variation, which would tend to reduce the magnitude of an effect (32). In our study prevalence of *H.pylori* infection was, 44.3% in the overweight group, and normal weight 35.8 %, but in the obese group it was 15.5% ?. Former smokers had high prevalence of 79.8 %, compared to current smokers, 5.5% and non-smoker 14.2%.Former alcohol consumer 79.8% and current alcohol consumer, 5.5 % .In a study from 17 geographically defined populations in Europe, North Africa, North America, and Japan, using a common protocol for blood collection and serological testing, found no effect of smoking or alcohol consumption on the prevalence of infection (32).. Education level also had marked difference in prevalence, group with primary education, 36.6 %,group with secondary education, 17.3%, group with no education level, 18.4%, and with higher education, 27 .9 % respectively. This is in conformity other studies which have found that low level of education standard was consistently and positively associated with the prevalence of infection. (32).

This study has its limitations: Former smokers and former alcohol consumers have association between *H.pylori* infections, but study lacks information on the quantity and duration of smoking and alcohol consumption? In our study there is association between smoking and alcohol drinking. Other researchers found no association with smoking and alcohol consumption (27,33).overweight subjects has association with *H.pylori* infection {44.3 %) but in this study obese subjects has *H.pylori* infection 15.5 %?.Future studies may able to address the routes and sources of transmission of *H.pylori* infection and some of the questions rose in this study.

CONCLUSIONS

The study reveals highest prevalence of *H.pylori* infection among in females and ethnic Kadazan, Dusun community. The socioeconomic factors, smoking, alcohol consumption and lower level of education were important factors in *H.pylori* infection. The *H.pylori* screening plan and eradication in high risk community will probably reduce the risk of cancer in the local population.

Authors Contribution

MM developed and wrote the manuscript .MJ critically appraised with suggestions.

Conflict of Interests

Authors have no conflict of interests.

ACKNOWLEDGEMENTS

The authors wish to thank Director General Ministry of health, Vice Chancellor, Dean school of Medicine University Malaysia Sabah and Director, Health Services Sabah for permission to publish this paper. We also express our appreciation to Directors Kudat,, KotaMarudu hospital, Luyang Clinic and Doctors and nurses of Queen Elizabeth hospital for their assistance. Financial assistance of Sabah State Government and Ministry of Science Technology and Innovation (MOSTI) is gratefully acknowledged. Ministry of Health Malaysia NMRR (**IDN0:0-976-6832).**

REFERENCES

1. Warren JR.(1983). Unidentified curved bacilli on gastric epithelium in active chronic gastritis. *Lancet*. **1**:1273-1278.
2. Marshall BJ.,Warren JR.(1984). Unidentified curved bacilli on gastric epithelium in active chronic gastritis. *Lancet*.**1**:1311-1314.

3. Cockburn M, Cox B.(1997). The effect of measurement error in the determination of Helicobacter pylori prevalence. *Epidemolo*gy.**8**:205-209.

4. Graham DY.(1999). Public health issues relating to *Helicobacter pylori* infection and global eradication. In: Graham DY, GentaRM, Dixon MF, eds. Gastritis. Philadelphia: Lippincott Williams & Wilkins 241-246.

5. Parsonnet J.(1983).*Helicobacter pylori*: the size of the problem. *Gut* .**43**:6S-9S.

6. NIH Consensus Conference.(1994).*Helicobacter pylori* in Peptic Ulcer Disease. *JAMA*.**272**:65-69.

7. International Agency for Research on Cancer. IARC Monographs on the Evaluation of Cancer Risks to Humans. vol. 61. Schistosomes, Liver Flukes and *Helicobacter pylori*. Lyon: International Agency for Research on Cancer, 1994.

8. Nomura A. Stomach cancer.(1996). In: Schottenfield D, Fraumeni JF, eds. Cancer Epidemiology and Prevention. 2nded. New York: Oxford University Press, 707-704.

9. Kuipers EJ, Thijis JC, Fasten HPM.(1995).The prevalence of *Helicobacter pylori* in peptic ulcer disease. *Aliment PharamacolTher*.9(Suppl. 2): 59-69.

10. Forman D.(1998). Lessons from ongoing intervention studies. In: Hunt RH, Tytgat GNJ, eds. *Helicobacter pylori*: Basic Mechanisms to Clinical Cure 1998. Dordrech: KluwerAcademic Publishers, 354-361.

11. Haruma K, Kamato K, KamadaT,*et al*..(2000). Helicobacter pylori infection is a major risk factor for gastric carcinoma in young patients. *Scand J Gastroenterol*.**35:** 255-9.

12. Moraes MMC, da Silva GAP.(2003) Risk factors for *Helicobacter pylori* infection in children. *J Pediatr*.**79**:21-8.

13. Rodrigues MN, Queiroz DM, BezerraFilho JG, *et al*.(2004). Prevalence of Helicobacter pylori infection in children from urban community in north-east Brazil and risk factors for infection. *Eur J gastroenterol Hepatol*.16:201-5

14. Van der HulstRWM, Tytgat GNJ(1996)..*Helicobacter pylori* and peptic ulcer disease. *Scand J Gasteroenterol*.31(suppl 220):10-8.

15. PersonnetJ, FriedmanGD, VandersteenDP,*et al*(1991*).Helicobacter pylori* infection and the risk of gastric carcinoma.*NEngl J Med*.**325**:1127-31.

16. Telford JL,Covacci A, GhiaraP,Montecucco C, Rappuoli R(1994)..Unravelling the pathogenic role of *Helicibacter pylori* in peptic ulcer: potential new therapies and vaccines.Trend Biochem.**12**:420-6

17. GohKL, Paraskthi N(2001).The racial cohorpehenomenon:seroepidemiology of *Helicobacter pylori* infection in a multi-racial South-East Asian country .*Eur JGastroenterolo Hepatol*.**13**(2):177-83.

18. UyubAM, RajSM, VisvanathanR,*et al.(*1994).Helicobacter pylori infection in north –east peninsular Malaysia: evidence for unusually low prevalence. *Scand JGasteroenterol*.**29**:209-213.(ref.no.18).

19. Rothenbacher D, Brenner H.(2003) Burden of *Helicobacter pylori* and *H.pylori*-related disease in developed countries: recent development and future implications. *Microbes Infect.*693-703.

20. Frenck RW Jr, Clemens J.(2003)*Helicobacter* in the developing world. *Microbes Infect*.**5**: 705-13.

21. Ertem D, Harmanchi H, Pehlivanoglu E.(2003).*Helicobacter pylori* infection in Turkish preschool and school children. Role of socio-economic factors and breast feeding. *Turk J Pediatr*.**45**:114-22.

22. Roosendaal R, Kuipers EJ, BuitenwerfJ,*et al*.(1997). *Helicobacter pylori* and the birth cohort effect: evidence of a continuous decrease of infection rates in childhood. *Am J Gastroenterol*.**92**: 1480-2.

23. Replogle ML, Glaser SL, Hiatt RA, *et al*.(1995) Biologic sex as a risk factor for *Helicobacter pylori* infection in healthy young adults. *Am J Epidemiol*.**142**: 856-63.

24. Perez-Perez GI, Bodhiatta L, Wongsrichanalai J, *et al*.(1990). Seroprevalence of *Helicobacter pylori* infections in Thailand. *J Infect Dis.***161**:1237-1241.

25. Dooley CP, Fitzgibbons PI, Cohen H, *et al* (!989). Prevalence of *Helicobacter pylori* infection and histologic gastritis in asymptomatic persons. *N Engl J Med*.**32**1:1562-1566.

26. Everhart JE, Kruszon-Moran D, Perez-Perez GI, *et al*.(2000). Seroprevalence and ethnics differences in *Helicobacter pylori* infection among adults in the United States. *J Infect Dis.***181:1359**-1963.

27. Graham DY, Malaty HM, Evans DG, *et al*. Epidemiology of *Helicobacter pylori* in an asymptomatic population in the United States.. *Gastroenterology.* 1991**:100**:1495-1501.

28. Parsonnet J.(!995) The incidence of *Helicobacter pylori*infection.*AlimmentPhamacolTher*. **9:45**-51.

29. Malaty HM, Grham DY.(1994) Importance of childhood socio-economic status on the current prevalence of *Helicobacter pylori* infection. *Gut.* **35**:742-5.

30. Webb PM, Knight T, Greaves S, Wilson A*et al.*(1994), Relation between infection with *Helicobacter pylori* and living conditions in childhood: evidence for person to person transmission in early life. *BMJ*.**308**:750-3.

31. Parkinson AJ, Gold BD, Bulkow L, *et al*.(2000). High prevalence of *Helicobacter pylori* in the Alaska Native population and association with low serum ferritin levels in young adults. *ClinDiagn Lab Immunol*. 885-8.

32. EUROGAST study group.(1993).. Epidemiology of and risk factors for *Helicobacter pylori* infection among 3194 asymptomatic subjects in 17 populations. *Gut.* **34**;1672-1676

33. Hook-Nikanne J.(1991). Effect of alcohol consumption on the risk of *Helicobacter pylori* infection.*Digestion*.**50**:92-8.